ACUTE DILATATION OF THE STOMACH:

ITS ETIOLOGY AND PATHOLOGY.

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Acute dilatation of the stomach is a condition of grave importance, which, if one may judge from the numerous cases which have been reported in recent years, is of much more frequent occurrence than has hitherto been imagined. Attention was first prominently drawn to it in this country, and its nature accurately described, by Hilton Fagge, who published in the Guy's Hospital Reports 1872-1873 two cases, which had come under his own care. Since that time considerable interest has been taken in the condition in England, Germany, and America, especially by Campbell Thomson, Ewart, Albrecht and Appel; and it is mainly to their enquiries that we owe our present knowledge of its nature. It has been possible to collect particulars of 61 cases (1-43), most of which have proved fatal although several complete recoveries have been recorded: the excessive mortality has probably been the determining factor in the publication of many of the cases, and one is inclined to think that many instances of a less severe type may occur without being correctly diagnosed. In so few instances has

a true diagnosis been made during life, and in these only after the dilatation has reached a very advanced stage, that the possibility of its existence is well worth bearing in mind when confronted with acute abdominal symptoms, especially if these arise in the course of an exhausting illness or after a severe surgical operation. The principal features of a typical case are a rapid increase in the size of the stomach, and the secretion of a large amount of greenish fluid together with acute and violent abdominal symptoms, followed by profound collapse, the case terminating usually in death.

Before proceeding to discuss in detail the etiology and pathology of the condition, and the numerous theories which have been advanced with regard to its causation, it is proposed to give a brief account of the symptoms and physical signs by which it may be recognised, and a description of the results which have been found when the abdomen has been examined after death. A knowledge of these facts is essential to a complete discussion of its origin. A detailed report of a case which was observed by the writer is given as an illustration, whilst a summary of the collected cases is appended at the end.

Symptoms and physical signs.

Although there are, in some particular instances, marked differences in the symptoms produced by acute gastric dilatation, yet there is a striking general uniformity which renders the disease a very characteristic one to describe. In most cases the onset is very sudden, and gives no warning whatsoever of its approach. Occasionally there have been vague gastric pains for some houre previously. The patient may be in excellent bodily health, or he may have become weakened by illness or operation, or he may have been recently the subject of some traumatism.

The first and most pronounced symptom is vomiting, which is violent and distressing: it tends to persist with, or without, slight interruptions. At times it has been relieved by treatment, only to recur whenever this was ceased. The vomit is usually greenish or brownish in colour, and in the later stages has been observed to be inky (Hood). The quantity is very large: bile is frequently present, and half-digested blood has been noticed.

There is severe pain in the epigastric region to which no relief is afforded by the vomiting. Great thirst is present as a rule, and hiccough is not uncommon: the state of the bowels varies;

although constipation is most frequently found, severe diarrhoea has been recorded, whilst in other cases normal movements of the bowels have taken place.

The pulse is rapid and thready, and the temperature may be subnormal. The urine is diminished in amount, and may be totally suppressed towards the end.

The expression is anxious: the face is pinched and the countenance sunken. The extremities are cold: cramps in the muscles and tetany have been present. Collapse quickly supervenes, and passes into coma, from which the patient does not often regain consciousness. In those cases in which recovery has occurred the symptoms have gradually subsided under treatment, and the patient has appeared to be well again in the course of two or three weeks.

On examining the abdomen one finds, on inspection, that it is distended unsymmetrically, the left hypochondrium being full whilst the right is flattened. The outline of the stomach is visible, and the lower limit of the greater curvature can often be made out. Schultz[37] has observed peristaltic waves in one case. Morris[33] has found the abdomen retracted, and in some instances the swelling

has disappeared after the use of the stomach tube. On palpation there is a feeling of resistance over the upper part of the abdomen. Fluctuation may be made out in the pubic region if much fluid be present, and succussion may be elicited on manipulation.

On percussion one ascertains that the normal resonant stomach note is obtained over a much larger area than usual, especially if the distension be due to gas. Dulness is present in the pubic region, its extent varying with the amount of fluid in the stomach. Localised areas of tenderness may exist in the epigastrium.

Morbid Anatomy.

The appearance of the stomach has been very well described by Thomson[39], and the writer is able to confirm his description. He says "it looks like a tightly distended cylinder, shaped like a V, the angle representing the lesser curvature, and being a very sharp one." On being removed from the body, the stomach shrinks and regains its customary size. The mucosa is swollen and injected, but often no microscopic changes have been evident. Pyloric obstruction has only been found in a few cases.

The duodenum frequently shares in the dilatation either in whole or in part. Dilatation of the stomach alone has been found in 26 cases; of the stomach and first part of the duodenum in 3 cases; of the stomach and first and second parts of the duodenum in 1 case; of the stomach and whole of the duodenum in 11 cases. The jejunum has been involved in 2 cases, whilst in one of Fagge's cases the caecum and ascending colon were also distended. The rest of the intestines are usually collapsed. Large amounts of fluid have been found in the stomach and even in the intestines; this is similar to that which has been vomited.

The superior mesenteric artery and its mesentery have been found to be tightly stretched over the commencement of the jejunum and to be exercising compression on it.

Autopsies were not obtained in four of the fatal cases.

Report of a case of acute gastric dilatation, which came under the writer's personal observation whilst Resident Medical Officer at the Royal Lancaster Infirmary.

The patient, a woman, aged 27, a domestic servant, was admitted to the Hospital on Feb. 16,

1904, under the care of Dr C. A. Rayne, with the history of an illness of several weeks duration. She had suffered for some time from severe pain in the region of the stomach immediately after taking food: this had been frequently followed by vomiting which gave her great relief. Once or twice she had had some haemorrhage from the stomach: she had lost weight rapidly.

When examined on admission she was found to be very emaciated, pale and anaemic, and looked extremely ill. The muscles were wasted, and the general nutrition was very poor. She complained of pain in the epigastrium after food, and frequent attacks of vomiting. The bowels were constipated: the tongue was furred, and her mouth and teeth were in a very septic state. The abdomen was not tumid and was not distended: there was tenderness on pressure over the epigastrium. The lower border of the stomach was found to reach as far as a point one inch above the umbilicus.

She complained of palpitation and breathlessness on exertion, and there was a soft systolic murmur audible in the mitral area. The blood was not examined. Gastric ulcer was diagnosed, and the patient put to bed on a milk diet, and ordered Bismuth Subcarbonate grs. XV. and Soda Bicarbonate

grs. V. every four hours. As the vomiting still continued, she was fed per rectum, only being allowed sips of water by the mouth. The vomiting ceased, and she began to look better. On the evening of Feb. 26, 1904, she was suddenly seized with violent pain in the stomach and an attack of severe vomiting. She vomited a large quantity of dark brownish fluid, and rapidly became collapsed. The upper part of the abdomen was distended, and there was distinct tenderness and resistance on palpation. Considering the acute nature of the abdominal symptoms and the previous diagnosis, a perforation of the stomach was feared, and arrangements were at once made to explore the abdomen. A catheter was passed and the bladder found to be empty: urine had not been voided for some hours. Ether was administered by Mr W. D. Barrow, and the abdomen opened by Mr A. S. Barling, assisted by the writer. An extremely dilated stomach at once presented when the peritoneum was incised. No signs of a perforation could be found. A tube was passed down the oesophagus, and 5 pints of a dark brown fluid were syphoned off. This seemed to contain partially digested blood. The cavity of the stomach was then washed out, and a posterior gastro-jejunostomy performed with a view to allowing the stomach contents to empty

themselves more readily. Towards the end of the operation the patient's condition became very grave, and recourse was had to the infusion of saline solution into the median-basilic vein. After a slight temporary improvement she died 12 hours after the operation, and 15 hours after the onset of the acute symptoms.

At the postmortem examination the stomach was found to be extremely dilated, and filled practically the whole of the abdominal cavity. The first part of the duodenum was also dilated. The rest of the bowel was collapsed. The remains of an old ulcer were found on the posterior wall of the stomach: there was no pyloric obstruction. The secretion of fluid had continued, as some quantity was found in the stomach and had not passed through the new opening, which was quite patent. No changes were evident in the gastric or intestinal mucosae. The mitral valve was incompetent. The kidneys and other organs appeared to be healthy.

This case corresponds very closely to the generally acknowledged type of the disease, and shows many features which are of great interest in view of its supposed causation. There was no previous error of diet, as the patient was at the time being fed by the bowel: there was no injury

or acute illness. She was exhausted by a long sickness of some weeks' duration. The vomiting was severe, and the amount of fluid vomited was very large. There was a total absence of foul gas: the nervous symptoms were slight, consciousness being retained. The condition was incorrectly diagnosed, the previous history leading one to suspect an entirely different state. Surgical treatment was adopted; and in this respect it is worthy of notice that it seems to be the only case in which an attempt has been made to procure relief by means of a gastro-jejunostomy, although Mayo Robson suggested it as probably the most suitable means to adopt. Unfortunately this did not enable the already paralysed viscus to overcome its difficulty. The absence of any form of obstruction, either at the pylorus or lower down, is important, as also is that of changes in the mucosa.

Etiology.

The information at our command for making a detailed study of the etiology of the dilatation is somewhat meagre and indefinite, owing to the small amount of literature which we possess upon the subject, and the relatively few cases which have been recorded. Of the 61 cases which have been

collected, 48 have terminated fatally, this representing 77%.

Sex:- there is a striking uniformity in the frequency of its occurrence in the two sexes. Males were affected in 29 cases, and females in 28.

Age:- only one case has been recorded below the age of 15, and that occurred in a child of 3. Whilst chronic dilatation is frequently observed in infants as a result of ill-feeding, acute cases have not been noted. Only 8 cases have occurred above 45, the oldest patient being a woman of 69. The decade in which most cases have been found is from 20-30, these numbering 19, 11 males and 8 females.

The following table shows the different ages:

	Male	Female	Total
Under 15		1	1
15 - 20	4	4	8
20 - 25	6	3	9
25 - 30	5	5	10
30 - 35	2	1	3
35 - 40	2	4	6
40 - 45	3	2	5
45 - 50	1	1	2
50 - 55	1		1
55 - 60	1	1	2
Over 60		2	2
	25	24	49

It is thus seen that no age is exempt: it is rare in the early and late years of life, being most commonly found between the ages of 15 and 45,

and the greatest number between 15 and 30 (27 cases out of 49). Neither sex appears to be more liable to it at one age than the other.

Previous health.

The physical condition of the patient at the time of the onset of the dilatation has shown great differences, for, whereas the majority have been suffering for a greater or less time from some other malady, several have been previously enjoying perfect bodily health, and where death has taken place in such instances no evidence of any pre-existing disease has been found at the autopsy.

Long and exhausting illnesses and attacks of acute fevers must be acknowledged as powerful predisposing causes. Amongst those which have been noted are puerperal fever, typhoid, scarlet fever, acute rheumatism, acute pleuro-pneumonia, phthisis, empyema, anaemia, endocarditis, fatty degeneration of the heart and chorea. Other conditions found postmortem include nephritis, myocarditis, purulent pericarditis, peritonitis, suppurating thrombus in the left cavernous sinus, retroduodenal abscess, and tumour of the pylorus.

Traumatism:-

This seems to have been an exciting factor in 5 cases: in three there was a history of a blow on

the epigastrium, and in the remaining two the spine had been injured, in one the patient having sustained a gun-shot wound in the lower dorsal region, and in the other a fracture of the occipital bone with a dislocation of the 4th and 5th cervical vertebrae. In the former of these two spinal injuries the symptoms of acute dilatation did not appear for 34 days, although there was complete paraplegia from the first: in the other four cases the symptoms came on at once, and were attributed directly to the accident.

Chloroform anaesthesia:-

This was assigned as a cause of Schitzler's case.

Surgical operations:-

These have preceded 15 cases; the nature of the operations was as follows:-

1. Amputation of the thigh; symptoms immediate.
2. Excision of the hip one month previously; symptoms probably due to indiscretion of diet.
3. Excision of the knee; symptoms immediate.
4. Operation on elbow; symptoms in 5 days.
5. Removal of necrosed bone in foot; symptoms in one hour.

6. Amputation of mamma.

7. Abdominal hysterectomy; symptoms in 14 days.

8. Double salpingo-oophorectomy; symptoms immediate.

9. Nephrotomy; symptoms in a few hours.

10. Nephrectomy; symptoms immediate.

11. Cholecystotomy (3 cases).

12. Duodeno-choledochotomy; symptoms in 10 days.

13. Excision of gall bladder and part of the transverse colon; symptoms immediate.

Symptoms also developed after a herniotomy in a case quoted by Wade in the British Medical Journal 1881, Vol.ii. p.471. This is not included in the list of cases, owing to the small amount of particulars given.

In connection with these post-operative cases attention is at once attracted to the number which have followed abdominal operations, especially upon the gall bladder. The interval of time between the operation and the onset of the vomiting has varied considerably. In most cases the symptoms have come on at once or in a few hours, but in some not until after 5 or 6days, and even in one case a month: in the latter case there is a history of an error of diet immediately preceding the attack. The diseases

for which the operations have been undertaken do not appear to be capable of offering any predisposing factor in the production of the dilatation, except in the instances where there was disease of the pancreas or gall bladder, and where adhesions of the pylorus had been discovered.

Indiscretions of diet:-

The eating of large and bulky meals on an empty stomach or the ingestion of unsuitable food, seems to have acted as an exciting cause in no less than 13 cases, 7 of which subsequently recovered.

As a result of this enquiry into the antecedents of these individual cases, one finds that there are four groups of factors which may, either separately or together, have some influence in producing the dilatation. Some may act as direct exciting causes; whilst others, by weakening the muscular coats of the stomach, may prepare the way for these, and render the distension more easy of accomplishment by diminishing the resistance of the muscle-fibres to a sudden increase of intra-gastric pressure, or to the extra stress caused by the introduction of food of an unsuitable consistency. These four groups are (1) acute fevers and septic diseases (2) injury (3) surgical operations (4) indiscretions of diet. In what way these factors act remains to be discussed.

Causation:

Ever since the day when Hilton Fagge first published his personal observations on the condition, and bestowed upon it the name of acute paralytic distension, numerous theories have been enunciated, and advanced as definite explanations of its nature and causation. These have differed widely from one another, and not one of them has so far met with anything approaching general acceptance. After carefully considering the particular characters and features of the collected cases, and noting in each one the pathological changes and complications that have been present, one is obliged to admit that, although extensive enlargement of the stomach has been a uniform phenomenon, yet the involvement of the rest of the intestinal tract has varied within such wide limits that it would seem almost impossible to attribute any common cause to all cases. Whilst some theories are capable of explaining certain cases with apparent accuracy, there is not one which can be accepted as applicable to the whole series. It cannot be denied that some may act as potent secondary causes, although their powers, as primary factors, cannot be recognised.

The following is a list of the many causes to which the dilatation has been attributed:-

(1) A sudden failure of compensation in an abnormally large stomach.

(2) An overdistension by the excessive fluid secretion which is so often present.

(3) Distension by gases.

(4) A spasm of the pyloric sphincter.

(5) A kinking in the region of the pylorus.

(6) An acute obstruction caused by compression of the small intestine by the superior mesenteric artery and its mesentery.

(7) Pressure of the stomach on the part of the duodenum which ascends by the side of the second lumbar vertebra to end in the jejunum.

(8) Prolonged anaesthesia in post-operative cases.

(9) Absorption of toxines.

(10) A paralysis produced either reflexly or by local interference with the nerves of the stomach.

According to the majority of these theories the dilatation is the outcome of a rise of pressure within the cavity of the stomach, which has resulted from a sudden increase in its contents or from an obstruction to their outflow along the intestines. In the minority an actual paralysis of the muscular fibres in the coat of the organ is supposed to take place, and the distension to be secondary thereto. In no theory is a process of active dilatation advocated, and it is impossible to conceive of it

happening. There must necessarily be some increase of pressure from fluid or gas to bring about the distension, but the question one has to decide is whether this pressure is the primary, or merely a secondary, cause. It is evident that, in order to establish any of the more numerous theories, one must show proof of the existence of an actual obstruction to the discharge of the gastric contents. As none has been usually found post mortem, experiments have been conducted on the cadaver to demonstrate the possibility of its production, but it is uncertain if precisely similar conditions would exist during life, and the evidence is conflicting. With respect to the theory of a paralysis, one should be able to find some pathological change in the muscle fibres or nervous mechanism, or to prove by experiment the possibility of a reflex action occurring under similar circumstances. We now proceed to discuss these theories seriatim, with as little overlapping as possible.

Attention has been drawn by many writers, and especially by Riegel[44] to a condition of the stomach, which has been termed megastria or megalogastria in which the capacity of the organ is of unusually large dimensions, although in other respects the stomach is perfectly normal. Ewald[45] emphasises

the necessity of distinguishing between this condition and that of gastrectasia, the dilated stomach. In his opinion the former may lead to the latter, although it is not in itself pathological: he considers that serious dilatation may exist along with complete compensation, and that, in consequence, the individual may continue to live in ignorance of it until such time as a failure of compensation occurs. If the compensation should fail very suddenly, the symptoms would be acute, and to such a sudden failure he attributes "those cases of acute gastric distension which have been described in English literature, i.e. by Fagge". He draws a comparison with those recognised cases where individuals have been the subjects of highly developed valvular disease of the heart without being cognisant of its existence, until the compensatory hypertrophy of the ventricle has given way under undue strain with disastrous consequences. Such cases have usually been encountered in the course of acute fevers, influenza, anaemia and chlorosis, where frequently acute myocarditis and fatty degeneration and infiltration have been found to have produced great weakening of the cardiac muscle, but where at times it has been impossible to discover with the microscope any pathological alterations.

In the latter circumstance the cause has been attributed to the lowering of the muscle tonus or an interference with the muscular innervation, and by some writers to a change in the cardiac ganglia. The comparison is not a suitable one as here there is as a rule a definite cause of the dilatation:- a weakness and diminished power of resistance in the muscle. One has already mentioned that there is often a history of one of the above diseases preceding acute gastric dilatation, and at the autopsy there has been found pericarditis (Hood), myocarditis (Albu), fatty heart (Dickinson); but no definite microscopic changes have been noticed in the muscular coat although there has been swelling of the mucous membrane of the stomach (Valsalva) as well as of that of the duodenum (Frazer). In no instance could one say that the patients were the subjects of megalogastria, as this would not account for the numerous cases, nearly one half, in which the duodenum in varying proportions, and even the jejunum, has shared in the distension. Moreover no signs of compensatory hypertrophy have been discovered, the walls of the stomach being thinned; and after the organ has been removed from the body at the post mortem examination it has quickly shrunk to its usual size. The same has been recorded

where the stomach was punctured or emptied during life, and afterwards regained its previous state which has not been one of extraordinary size (Robson). As the eating of bulky meals by poorly nourished and starving persons seems to have acted as an exciting cause of dilatation, it is possible that this might occur all the more readily in a stomach that was abnormally large, or in a state of chronic enlargement; the symptoms due to the pressure of the loaded organ would arise more quickly. In the only case in which chronic dilatation was known to exist previously the symptoms came on after lavage, and there was found marked pyloric obstruction due to a growth (Thomson. Case I.), but in chronic cases there is no perceptible thickening of the walls, the fasciculi being stretched out and flattened.

As a proof of the possibility of the existence of dilatation unknown to the patient, Ewald quoted an instance in which he had inflated the stomachs of two old persons, who had not previously complained of any gastric disorder, and found in each well-developed dilatation. Although one is not prepared to agree with those French observers who regard dilatation as an event of such frequent occurrence, one certainly admits that the capacity of the stomach in different individuals does vary within a wide

range, and one acknowledges the existence in some cases of true megalogastria, but one cannot conceive of it affording an explanation of acute dilatation.

We now pass to study the fluid secretion, which is so often present in such excessive quantities, with a view to ascertaining what bearing it has upon the dilatation. Hilton Fagge regarded it as the primary cause, and thought that the stomach was "paralysed from overdistension and unable to rid itself of its burden". In this opinion he was supported by Morris[33], who introduced the name of "acute gastrorrhoea" as a more suitable term. Morris compared it with the paralytic secretion which was obtained by Moreau when a loop of intestine was isolated, and its nerves divided; but Pye Smith and Lauder Brunton[46] have shown that this secretion was the result of the removal of some small ganglia in the solar and superior mesenteric plexuses, and that it was uninfluenced by section of the vagi and splachnic nerves. Excessive secretion has been recorded in many other conditions as tabes, hysteria and neurasthenia[47], chronic dyspepsia[48] and chronic dilatation[49]. The nature of the secretion is not constant: the percentage of hydrochloric acid varies - it may be

normal or in excess. The cause in these cases seems to be an increased irritability either of the mucous membrane or of its secretory nerves. In many cases no anatomical changes have been found in the mucosa[50] just as in acute dilatation, which would seem to indicate a nervous origin. It is possible that a hypersecretion may be caused by a stimulation of the secretory centres of the brain, and this would compare with the salivation found in progressive bulbar paralysis. This could be understood if the cause was a toxaemia, and, as will be mentioned later, many cases show evidences of the existence of a toxic state.

The fluid is not invariably present as the stomach is often distended entirely by gas: in a case observed by Humby and Miller[32] the muscular fibres had given way in places, allowing the mucous coat to approximate to the peritoneal, and this was evidently due to the distending force of the fluid. Thomson[39] reports an early case in which no fluid was present. The secretion is not essential to the production of distension and cannot therefore be looked upon as the primary factor. One is inclined to recognise it as a paralytic secretion secondary to the dilatation, as there is good evidence that the walls of the stomach are first

paralysed and rendered incapable of expelling their contents, thus permitting the fluid to accumulate to such a large extent, just as is observed in retention of urine. The two conditions can occur quite independently of each other, but the excessive secretion will increase the dilatation if they occur together, the separation of the fibres in Humby's case affording convincing proof of this.

Owing to the frequency with which the stomach has been found to be tensely distended by gas, endeavours have been made to attribute the primary cause to a sudden and overwhelming evolution of gas, leading to a rapid and fatal cardiac failure. That the presence of such an amount of gas must be a very important factor, either primary or secondary, is evident, when one notices the intensity of the distension, and the relief afforded by its removal by puncture or the stomach-tube. It is present in greater or less quantities in all cases, whereas the fluid is often absent or very little.

Sir William Broadbent[9] has pointed out that bulky meals and large amounts of fluid will distend the stomach, and that this will be aggravated by the gases produced by the fermentation of saccharine and amylaceous matters. He considers that such a

dilatation may occur with an acute onset. In only one of the two cases observed by him (both of which recovered) was there is history of a hearty meal, the other having followed the eating of an ice on an empty stomach, but in this instance the symptoms came on during the following day and he concluded that the ice had paralysed the secretory and muscular apparatus, and that food had accumulated and fermented till the attack was provoked.

According to this the gas is to be regarded as a secondary cause.

Jacob[51] expressed the view that it might possibly be caused by gas evolved during imperfect digestion, the stomach walls being in an atrophic state from partial disuse in a patient who had been able to take only a small amount of food, or had been exhausted by a long illness. Clifford Allbutt[52] states that overdistension by gas occurs in some cases of gastralgia and remarks that there is a marked difference between this gas and that generated by the fermentation of food, as it is suddenly produced and is entirely innocent of odour or taste.

Ewart[53] regards acute gaseous dilatation as an incident of some frequency, though not often fatal: he does not look upon it as a result of any

permanent obstruction as the symptoms are relieved by vomiting. He attributes the pain to excessive gastric inflation.

Herringham[54] and Pavy[55] both think that the gas is a secondary factor in the causation.

The bulk of the gas present has been said to consist of air that has been swallowed, whereas the gases produced by fermentation are Carbon Dioxide, Hydrogen, and Methane[56]: for a favourable production of these the absence of hydrochloric acid is desirable, although Naunyn[57] says there may be fermentation when there is no diminution in the secretion of acid or in the motility of the stomach. The fermentation may then be due to the introduction of excessive amounts of fermentable material together with the agents which cause the action.

Krehl[58] thinks that the products of fermentation may irritate and injure the gastric mucosa, giving rise to pain and vomiting, and possibly a spasmodic stricture of the pylorus with diminished gastric motility.

Unfortunately the fluid and gas have not been examined in very many instances. There is great need for this to be done in cases that are met with in the future, as much information may thereby be obtained. Humby and Miller[32] state that they

found numerous sarcinae ventriculi in the vomit of their case, which would indicate some degree of fermentation. Indiscretions of diet have been the exciting cause in 12 cases and the association of the symptoms with heavy meals lends considerable support to the theory of distension by gas. Fermentation by irritating the mucosa might be the origin of the increased secretion (vide supra). The true source of the gas is very difficult to explain: gaseous distension of the intestines after surgical operations, especially on the intestinal tract itself, is a well-known and troublesome phenomenon. The cause has been recognised as a paralysis of the muscular coats, and one has found the benefit to be derived from the exhibition of drugs which stimulate the peristaltic contractions. The introduction of a tube into the rectum allows of the escape of the imprisoned gas, and relieves the distension, diminishing the adverse pressure and making peristalsis easier.

One has recently had an opportunity of examining a little girl, aged 9 years, an imbecile, who was under the care of Dr W. H. Coupland of the Royal Albert Asylum at Lancaster, and who suffered from the very uncommon condition of congenital dilatation of the colon. She was subject to repeated attacks of acute abdominal symptoms with extreme collapse,

and distension of the abdomen. Relief was always obtained by administering saline enemata. There was a large accumulation of flatus, and constipation was always most troublesome, as if neglected an acute attack was provoked.

Kemp[27] mentions two cases of acute gaseous distension and attributes them to errors of diet. In one the patient has 20 attacks in 10 years: emesis gave her relief. He compares them with the temporary distension produced in children by over-feeding, and in which emesis is of great benefit.

So many cases of chronic dilatation have been proved to have been dependent upon an obstruction at the pylorus from either a cicatricial contraction or a new growth, that many writers have urged that the acute cases may be due to a spadmodic closure of the pylorus which is set up by irritation either of the vagus nerve, or of the opening itself by the contents of the cavity. Lauder Brunton[59] has inclined to the former view, whilst the latter was suggested by Pepper and Stengel. Both are difficult to uphold, and it becomes necessary now to consider the normal mode of action of the sphincter pylori. The stomach receives its nerve supply from the vagi and the splachnics between which there are

very free communications. These extrinsic nerves contain efferent fibres, motor and secretory, and afferent fibres to the central nervous system. It has been proved that the efferent fibres of the vagus are motor, and that artificial stimulation of them produces contractions of the muscle fibres of the stomach; whilst those of the sympathetic are inhibitory and stimulation of them produces a dilatation of the stomach and relaxation of the sphincter. The mechanism controlling the latter action during digestion is obscure. It has been connected with the consistence of the food, and has been said to be regulated by chemical stimuli originating in the food. Normally it appears to have some relation with the presence of free hydrochloric acid: the acid is neutralised in the duodenum by the alkaline secretion of the pancreas, and as this is done more of the gastric contents are allowed to escape through the orifice. Here it may be remarked that no definite relationship has been established between the motility of the organ and the acidity of its contents. The consistence of the latter is of great importance as large particles are not allowed to pass through the orifice but are thrust back into the fundus: this seems to be the work of the pyloric antrum. Warm materials are found to relax

the sphincter[60] and one wonders if cold could have the opposite effect, as in one of Broadbent's cases the symptoms supervened on the eating of an ice on an empty stomach. Cannon and Murphy's experiments[61], to be considered later, incline one to the possibility of pyloric spasm, and one has already spoken of Krehl's suggestion regarding the consequences of fermentation.

Sanctuary[62] has described a case of dilatation with a patent sphincter where there was above the opening an oval ulcer which he thought has set up irritation resulting in a violent contraction of the pylorus. Hyperacidity exists so often that one would expect to find cases much more commonly if it alone could produce dilatation; but Rudimeyer also thinks hyperacidity may produce irritation of the pylorus. See[64] finds a definite causal relationship between hyperacidity and atony of the stomach, but the cases described by him are not acute ones.

The great arguments against the existence of any stricture are the presence of bile in the vomit, and the frequency of involvement of parts of the duodenum and jejunum, but the latter might result from an interchange or varying distribution of nerve fibres.

Ochsner[64] states that, in many cases at operations, the duodenum is distended with gas to a point

just below the entrance of the common duct, whilst below this it is contracted, as is also the origin of the jejunum. Dilatation of the upper part of the duodenum was most commonly present in patients with chronic cholecystitis with sand or gall-stones in the gall bladder. In these cases there was more or less enlargement of the pancreas. In the vomit examined after anaesthesia bile was invariably present, showing there must be some reason why this fluid should be forced upward past the sphincter pylori rather than downwards through the small intestine: the pain was relieved by lavage and prohibition of all food. In a series of cases examined there was a greater or less degree of narrowing between the pylorus and entrance of the common duct, and more or less thickening of the wall, 2 to 4 cm. below the duct, the circular fibres being markedly increased, forming a species of sphincter. Ochsner concludes that under certain conditions of the gall bladder this duodenal sphincter may obstruct the passage of food and gas. In five cases of acute dilatation the attack followed an operation on the gall bladder, but in all these there is no mention of duodenal involvement. A spasmodic stricture of this sphincter would explain these cases, where a similar affection of the pylorus could not, owing

to the presence of bile in the vomit: it could also be applied to those cases where the 1st and 2nd parts of the duodenum are involved, but will not explain implication of the jejunum.

Cahn[65] has described a case due to pyloric stenosis with ampulliform dilatation of the duodenum; and ballooning of the rectum below a stricture is a well-known phenomenon.

In the face of all this evidence one does not feel able to deny that a pyloric spasm may exist, as well as one lower down in the duodenum and some cases may really be dependent on such a cause. Peristaltic waves have only been seen in one case (Schultz), which speaks against it, but one must not forget that to produce these the muscular and nervous apparatus must be fairly sound and they are probably paralysed.

The possibility of a kinking in the region of the pylorus has been favoured by Bradshaw[12] because he thinks that without obstruction the fluid would have escaped into the intestines and not have been vomited, and because of the collapse of the bowels. Fleiner[66] also attributes to this cause those cases following laparotomy.

Ewald states that cicatricial bands may compress the duodenum or press it against the posterior

abdominal wall, so producing traction and flexion of the pylorus or the horizontal part of the duodenum.

Rokitansky[67] has observed dilatation giving rise to traction and dislocation of the organ, and probably flexion of the duodenum. Kussmaul[68], experimenting on animals, has found that, when the abdominal walls are greatly relaxed, rotation of the distended stomach has caused the pylorus to take an antero-posterior position, and at the same time to twist and compress the pars horizontalis duodeni at its point of departure from the stomach in such a way that not a drop of fluid can pass through the opening. When the stomach is full and its ligaments lax so that it drags this part of the duodenum down with it, the lumen of the bowel may be occluded by a kink, not at the pylorus, but lower down at the junction of the horizontal and descending parts of the duodenum. In Wiesinger's[42] case the laparotomy revealed the stomach twisted at an angle of 180° at the pylorus, and recovery followed upon puncture and release of the gas: in Hughes Bennett's[4] case there was rotation at the junction of the cardia and the oesophagus. Thomson[39] also noticed some rotation in one case. One looks upon this rotation as due to the distension, and a study

of the anatomical position of a distended stomach shows how a kinking could arise. The long axis becomes directed more obliquely downwards and to the right from the fundus: the pyloric part moves towards the right, and is checked by the duodenum: the stomach is curved at the pylorus, the antrum forming the centre of the bend and the pyloric canal being directed backwards and upwards. The mobility of the 1st part of the duodenum which alone receives a complete covering of peritoneum prevents any kinking, but the presence of adhesions might permit it and in one of Robson's cases there were many present. The curve of the duodenum becomes U-shaped, and Quain says it may even cross the 3rd lumbar vertebra with a sharp V-shaped curve.

The 3rd part of the duodenum is firmly fixed by the musculus suspensorius duodeni which arises from the left crus of the diaphragm, and it is crossed by the superior mesenteric vessels: it is also related anteriorly to the stomach. The dilatation has been put down to pressure by these vessels or by the stomach itself. Albrecht[1] found the dilatation ceasing at the level of these vessels, and quoted a case of Schnitzler's where the artery was observed to be constricting the commencement of the jejunum. Ewart[53] considered that the collapsed

intestine drags the mesentery and artery as a cord over it, and he suggested as predisposing causes a congenitally long mesentery plus malnutrition and exhaustion. The position of the line of demarcation between the distended and the collapsed parts of the bowel is not a constant one, nor are the intestines always collapsed. Constipation is not an invariable feature as diarrhoea has been noted (Box and Wallace) and the bowel has been full of fluid (Frazer). Hence this cannot be accepted as the cause.

Box and Wallace[6] distended the stomach with water on the cadaver and found that it could be fully distended with the jejunum cut across, even when the superior mesenteric vessels and their mesentery were divided. By introducing the finger behind the stomach and raising the fundus and posterior wall, it was possible to allow the fluid to escape through the divided end. When the duodenum to the right of the spine was cut across, no distension could be produced. They concluded that the obstruction was due to pressure of the stomach on the part of the duodenum which crosses the 3rd lumbar vertebra.

Out of the 61 collected cases 14 have occurred subsequent to severe operations. Albrecht thinks

that they may be due to prolonged anaesthesia. Henry Morris was of the opinion that the anaesthetic could not be blamed. Cannon and Murphy[61] have made an exhausted study of the movements of the stomach and intestines in certain surgical conditions, and have found that under normal circumstances food begins to leave the organ in 10 minutes after ingestion and that it is empty in 3 hours. After high intestinal operation (18 cm. below the pylorus) food begins to emerge only after 5 or 6 hours. They consider that the delay is due to an altered functioning of the pyloric sphincter and not to the administration of ether. The gastric peristaltic waves were seen running continuously so long as any food remained in the stomach. In post-operative cases, after the first half hour of digestion they observed the waves travelling regularly towards the pylorus, but the sphincter held tightly closed against them. They found that the time this lasted compares with the time required for primary cementing of intestinal wounds which is usually 6 hours[69]. There was no question of the patency of the lumen of the gut. They could not determine the cause of the closure of the pylorus, but suggested that the injured intestine might be the origin of a reflex action on the sphincter which is mediated through the central nervous system or through the local

nervous mechanism of the intestinal wall. They concluded that the effect of ether was to delay the initial passage of food from the stomach for a short time and to slow the rate of discharge, that it did not produce an inactivity but merely a slowing of the movement of the food through the canal. The description of this action of the pylorus is especially interesting in view of the question of spasmodic stricture. Cannon and Murphy also found that in cases of thrombosis and embolism produced by tying the arteries, in only one of 6 cases was there any activity of the stomach and intestines. In 5 cases postmortem the gut was found to be empty below the affected part. They state that in these experiments care was taken to avoid including any nerves in the ligatures.

Kemp[27] considers the post-operative cases to depend on some shock to the sympathetic system although he thinks that sometimes it may be due to the anaesthetic or to uraemia.

Embley[70] has demonstrated that reflex cardiac inhibition may be produced from the vagus by chloroform and it is feasible that the same might result with the stomach. The interval between the operation and the onset of the symptoms has varied from a few hours to several days, in one case even to a

month (Jessop) so that one would expect that all the effects of the anaesthetic would have had ample time to pass off. Most of the patients would be considered as fit subjects to bear the administration of a general anaesthetic, although in one case there was nephritis, (Thomson, Case I.) and in another pulmonary oedema (Goodhart[22]): in each of these instances vomiting commenced soon after the operation.

Robson[35] had two cases of recovery in post-operative dilatation, and it is possible that many cases of severe post-operative vomiting with collapse may be examples of a less severe type of the disease, due to such an altered function of the pylorus as Cannon and Murphy describe. Their theory of the injured bowel being the site of origin of a reflex inhibition is one worthy of careful investigation, as if correct it would also explain those cases where there has been some injury to the abdomen and its contents. The influence of the anaesthetic seems to be of no account in the causation.

Having failed to secure a satisfactory solution by means of the theories of obstruction, or distension by fluid or gas, one turns to the question of the existence of a primary paralysis of the muscular fibres of the stomach wall, and one recalls

the term of "acute paralytic distension" suggested by Fagge, who regarded the vomiting as ceasing because of a paralysis of the muscle-fibres.

Goodhart[22] concluded from an analysis of all the cases of dilated stomach, not due to pyloric obstruction, observed at Guy's Hospital from 1875-1882, that paralysis of the viscus is, "if not the determining cause, at any rate an accompanying condition."

The causes which might lead to muscular insufficiency may be enumerated as follows:-

(1) Ingestion of excess of food or drink, setting up an acute gastritis.

(2) Acute and exhausting illnesses.

(3) Renal or cardiac disease producing oedema of the gastric walls.

(4) Adhesions restraining the movements.

(5) Disease of the nerves or interference with their trunks or branches.

(6) The action of toxines on the nerve or muscle fibres.

In many instances there has been a history of the first three factors, but, as we have seen, the changes in the walls are very uncertain and seem to be very slight. Laryngeal catarrh is known to produce a paralysis of the muscles of the vocal cords and a gastric catarrh may conceivably have the same

effect. Constipation has been a troublesome antecedent, and Sée and Mathieu[80] have pointed out that a permanent sluggishness or paresis of the intestines might cause a diminution of the gastric peristalsis. Broadbent thought one of his cases depended on paralysis from eating an ice, and ether has been seen to bring about a slowing of the rate of discharge from the stomach. A somewhat similar condition is found in acute paralytic ileus which is met with in hysterical patients, in the course of diseases of the nervous system and after blows on the abdomen.

Andral has described a case of dilatation where there was no obstruction, yet well marked ulceration, and has explained it as a paralysis of the pyloric region following the destruction of the branches of the vagus.

Acute oesophageal dilatation has taken place where there was no demonstrable stenosis, and it has been assigned to a primary paralysis of the muscular coat, probably consequent on a lesion of the vagus. Krehl[71] has produced a similar result in a dog by dividing the vagi in the neck; and Carion and Hallion[72] have obtained dilatation of the stomach and part of the oesophagus by a similar experiment. Wertheimer[73] obtained reflex inhibition of the

tonus of the stomach by stimulation of the central end of the vagus.

Pawlow[74] proved that the secretion of the gastric juice is controlled by the nervous system, and that the secretory fibres are contained in the vagi, but it has been said that digestion can continue both as regards movements and secretion when the nerves are divided[75].

One can well understand that there might be some local interference with the nerves in many of the recorded cases, especially in those following operations on the gall bladder and intestines, and in those occurring in pleuro-pneumonia, empyema and other lung diseases, but no definite lesions have been discovered. One recalls, however, Cannon's suggestion of a reflex inhibition.

With the present day tendency to ascribe nearly every difficult problem to a toxaemic state, it is not surprising that this has been proposed to solve this question. It has been suggested by Hoffman that the stomach is essentially an automatic organ, and that its movements are merely regulated by its extrinsic nerves, but no proof is forthcoming as to whether such automatic property is possessed by the muscle fibres or by the intrinsic nerves -

the plexuses of Auerbach and Meissner. It may be that this power is paralysed by the action of toxines absorbed from the gastric contents or that they might exert an influence through the central nervous system. Clifford Allbutt[76] thinks that in the acute fevers the toxic state of the blood "may intensify, if it do not set up, the mischief", and compares it with cases where the heart dilates quite apart from endocarditis or pericarditis. Nervous symptoms have been very marked on some occasions. Broadbent[9] describes severe tetany in one case, and this is a well-known and fatal occurrence in chronic dilatation. Gerhardt[77] refers it to the absorption of toxines, and Minkowski evidences cases where coma has been produced by auto-intoxication. Howard[78] inclines to toxaemia as a cause of the tetany and says the intoxication may be exogenous, but is more often autogenous and produced in the stomach.

Bonveret and Devic prepared extracts from the stomach contents, and injected them into animals, producing nervous phenomena.

Kussmaul[79] assigns the tetany to the sudden decrease of water in the tissues, as in cholera, and one notes that tetany occurred in a case that had been treated by lavage and where the production

and absorption of toxines would be thought to be very difficult.

Kemp[27] mentions that Mengelsdorf who examined 400 cases of migraine frequently found acute gastric dilatation. Lauder Brunton[59] also found the same.

In the present state of our knowledge one cannot express any opinion on the possibility of the action of toxines, and one is inclined to consider the tetany to depend on the loss of fluid in the tissues, and the collapse to heart failure and exhaustion from the vomiting. The severe vomiting has been brought forward as an argument against paralysis, but Box and Wallace showed that vomiting can be produced by injecting tartar emetic even when the stomach has been replaced by a bladder, provided there is a good communication with the oesophagus, and the abdominal muscles can contract properly.

In order to establish a satisfactory solution of the subject under discussion, it is evident that there are three factors which call for careful study; these are (1) the gastric contents (2) the action of the muscular coat (3) the resistance offered by the pylorus. If the relationship, which normally exists in health between these factors, be in any way altered, dilatation will be the natural outcome. One has considered the nature and origin of the excessive

secretion and the accumulation of gases, and one concludes that, as neither of them are constantly present, they cannot be essential to the production of dilatation. Obstruction at the pylorus or a lower position in the intestinal canal has been observed in so few instances that the desired solution cannot be obtained from it. One is left then with the second factor and one looks upon this as the primary defaulter and regards the cause as a paralysis of the muscular coat. The influence of many secondary factors is by no means to be ignored and the dilatation will be aggravated by anything unusual in the consistence of the food, and still further by an undue resistance to its outflow, whether that be produced by a spasmodic stricture of the pylorus, or by an obstruction owing its origin to a kinking, rotation or compression of the duodenum.

A P P E N D I X.

1. Extracts of 61 collected cases of Acute Dilatation of the Stomach.

2. Analysis of above cases.

3. References.

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EXTRACTS OF 61 COLLECTED CASES OF ACUTE DILATATION OF THE STOMACH.

1. Albrecht[1].

Woman, aged 44, with cancer of mamma. After amputation of breast she had acute abdominal symptoms, persistent vomiting and collapse. Death two days later.

Post Mortem: Extreme dilatation of stomach and duodenum, as far as level of superior mesenteric artery.

2. Albrecht.

Woman, aged 30 : operation on elbow : persistent vomiting five days later : marked constipation : death in eleven days.

Post Mortem : Extreme dilatation of stomach.

3. Albu.[2]

Man, aged 26 : had scarlet fever. Seized with epigastric pain, and incessant profuse vomiting for 24 hours. Abdomen distended : fluctuation and succussion obtained. Death in two days.

Post Mortem : Stomach enormously distended. There was no pyloric obstruction. Mucosa swollen and discoloured : signs of myocarditis and nephritis.

4. Appel:[3]

Abdominal injury followed by dilatation of stomach. Laparotomy, stomach drained and closed up. Dilatation recurred.

Post Mortem: Stomach dilated.

5. Bennett:[4]

Man, aged 26, with empyema. Sudden pain in abdomen: vomiting and distension. Death in nine days.

Post Mortem: Stomach distended with air.

6. Bettmann:[5]

Girl, aged 17, with typhoid: vomiting, delirium, abdominal pain, distension of abdomen, loss of liver dulness and collapse. Lavage practiced. Recovery occurred slowly.

7. Box & Wallace:[6]

Boy, aged 16, blow on epigastrium, followed by vomiting and collapse. Distension of abdomen. Laparotomy. Stomach incised and drained. Death in a few hours.

Post Mortem: Stomach dilated.

8. Box & Wallace:

Man, aged 24, with pneumonia. Had profuse diarrhoea on eighth day : on tenth day severe vomiting and collapse. Death on eleventh day.

Post Mortem: Stomach and duodenum dilated.

9. Box & Wallace:

Man, aged 29, amputation of thigh, followed by collapse and death.

Post Mortem: Stomach and duodenum dilated; the latter as far as the front of the 3rd lumbar vertebra.

10. Boas:(7)

Patient, aged 20: dilatation following error of diet. Recovery.

11. Broadbent Walter:(8)

Man: abdominal pain and vomiting after eating heavy meal of pork on empty stomach. Death in two days.

Post Mortem: Stomach enormously distended.

12. Broadbent Sir Wm:(9)

Woman, aged 35: took an ice on an empty stomach. The next day had violent epigastric pain with severe tetany in the hands and feet. She vomited after some brandy and lost consciousness. Stomach was dilated. Recovery occurred.

13. Broadbent Sir Wm:

Man, aged 50, ate a hearty meal on a channel steamer: was not seasick. After landing he became faint and vomited copiously. Stomach was dilated. Recovery complete.

14. Brown H. H.[10]

Girl, aged 16, with acute rheumatism and chorea: had sudden abdominal pain and vomiting with collapse: perforated typhoid ulcer suspected. Death in 12 hours.

Post Mortem: Stomach dilated and full of greenish brown fluid: colour due to acid haematin. Some brown patches in the mucosa: signs of recent endocarditis present.

15. Brown, W.H.[11]

Man, aged 55: symptoms of acute intestinal obstruction: intense pain and vomiting: hands cold, pulse thready, face pinched: abdomen distended above the pubes, flattened at the sides and epigastrium: Resonant except between the umbilicus and pubes, where it was dull: fluctuation and succussion were obtained. Aspiration performed: then laparotomy. A large cyst was found and three pints of greenish fluid removed.

Post Mortem: Cyst was found to be the stomach, very dilated: no obstruction found: marked nephritis.

16. Bradshaw:[12]

Woman, elderly: had three different attacks of sudden dilatation in all of which relief occurred.

17. Dickinson:[13]

Girl, aged 3: had fatty degeneration of heart.

Post Mortem: Stomach distended with gas and contained large lumps of food.

18. Dyson:[14]

Woman, aged 41: pregnant seven months: had a premature labour - after exertion : vomiting and death in three days.

Post Mortem: Not obtained.

19. Edmunds:[15]

Man, aged 35: had gunshot wound of lower dorsal spine: 34 days later had abdominal distension and collapse. Death in nine hours.

Post Mortem: Stomach full of gas and fluid: no obstruction.

20. Erdmann:[16]

Man, aged 18, had a fall backwards, followed by abdominal distension and pain. Recovery.

21. Fagge Hilton:[17]

Man, aged 30: with incipient phthisis: had persistent vomiting, anuria, gradual collapse, abdomen retracted, dulness half way up to umbilicus. Bladder empty. Succussion obtained.

Post Mortem: Stomach distended with fluid. When removed from the body it shrank to its natural size, showing on the surface striae like linae gravidarum.

22. Hilton Fagge:

Man, aged 20: had abdominal pain and vomiting for fourteen days: countenance sunken, eyes glassy, breath sweet, abdomen distended, right hypochondrium flat, succussion obtained. Stomach tube was passed and seven pints of greenish fluid removed. Death in four hours.

Post Mortem: There was a sloughing abscess behind the duodenum communicating with the bowel. Stomach was dilated but shrank on removal from body.

23. Fenger:[18]

Dilatation following five days after a cholecystotomy: death in another five days.

Post Mortem: Stomach dilated.

24. Fisher:[19]

Woman, aged 36. Death from suppurating thrombus of left cavernous sinus.

Post Mortem: Stomach and duodenum much dilated. No obstruction present.

25. Fisher:

Woman, aged 25. Death from puerperal fever.

Post Mortem: Stomach containing 8 oz. fluid extended below umbilicus: was capable of holding 150 oz. Duodenum also was double its normal size: jejunum unaffected.

26. Frazer:(20)

Man, aged 20, with pleuro-pneumonia. When apparently convalescent suddenly began to vomit greenish fluid. This continued for two days before he died.

Post Mortem: Dilatation of stomach and duodenum. Mucous membrane injected and blackened.

27. Fürstner:(21)

Woman, aged 21, received a blow on the epigastrium: had intense pain, convulsions, swelling of gastric region which subsided in four days.

Treatment: Ice, morphia, lavage and induced current. Swelling became reduced but recurred. Recurrence subsided under the current.

28. Goodhart:(22)

Man, aged 29. Excision of knee followed

by vomiting which persisted till death - 75 hours later.

Post Mortem: Stomach dilated.

29. Hood:(23)

Girl, aged 19, had some inflammatory mischief in right lower jaw, and some difficulty in respiration with pneumonic crepitations and a high temperature. Five days later had severe vomiting which persisted: vomit became inky. Death in 12 hours.

Post Mortem: Stomach extended below the umbilicus: duodenum and upper part of jejunum also dilated. No erosion of stomach or bowel. Both lungs were engorged and there was acute purulent pericarditis.

30. Hunter:(24)

Woman, aged 25. Both tubes and ovaries were removed. After the operation she had severe vomiting and death took place in eight days.

Post Mortem: Stomach was greatly dilated: no pyloric obstruction found.

31. Jessop:(25)

Woman, aged 26: recovering from an excision of hip performed a month previously for a condition of traumatic origin. She ate an apple

and had severe vomiting for two days. On third day stomach was distended greatly. It collapsed after vomiting. Abdomen opened and stomach, duodenum and first 6 in. of the jejunum were found to be dilated. Rest of bowel collapsed.

Post Mortem: Dilatation ceased abruptly 6 in. from the duodeno - jejunal flexure.

32. Kelyneck:(26)

Woman, aged 19: had severe pain in right hip due to tuberculous disease. Was seized with sudden vomiting of green fluid. Stomach was found to be dilated and was washed out. Vomiting persisted till death four days later.

Post Mortem: Right pleura adherent, stomach much dilated and also first part of duodenum. Intestines were collapsed below the superior mesenteric artery. No obstruction found.

33. Kemp:(27)

Dilatation occurred during Typhoid fever: intestines also distended with gas. Recovery.

34. Kemp:

Woman: dilatation followed overeating: she had 20 attacks in ten years.

35. Kemp:

Woman: dilatation occurred during an attack

of rheumatic endocarditis: tachycardia present, was supposed to be due to improper diet. Recovery occurred: treated with resorcin.

36. Kirch:[28]

Man, aged 19: acute abdominal symptoms followed a supper of soup and beer: diagnosis made. Succussion obtained. Death in two days.

37. Krundat:[29]

Man, aged 22: had acute abdominal symptoms: laparotomy performed. Death in two days.

Post Mortem: Stomach and duodenum dilated.

38. Krundat:

Man, with typhoid fever. Death two days after onset of acute symptoms.

Post Mortem: Stomach and duodenum dilated.

39. Krundat:

Woman, aged 69. Death 11 days after onset of acute symptoms.

Post Mortem: Stomach and duodenum dilated.

40. Lieutaud:[30]

Man. Death followed quickly after a meal. Abdomen was distended.

Post Mortem: Stomach dilated: lungs suppurating.

41. Meyer:(31)

Man, aged 41. Acute abdominal symptoms followed in 11 days by death.

Post Mortem: Stomach and duodenum dilated, the former compressing the third part of the latter.

42. Miller & Humby:(32)

Woman: aged 48, of nervous temperament: had sudden vomiting: death in ten days.

Post Mortem: Stomach dilated: muscular fibres had given way in places: Sarcinae found in vomit.

43. Morris:(33)

Man, 37. Had suppuration in right ankle-joint: operation followed by vomiting which persisted till death, 41 hours later.

Post Mortem: Stomach dilated : submucosa injected.

44. Morris:

Man: acute dilatation followed upon nephrectomy. No autopsy.

45. Munro:

Woman, aged 27. Acute symptoms during long illness. Laparotomy revealed greatly dilated stomach. Gastro-enterostomy performed. Death.

Post Mortem: Stomach and first part of duodenum dilated.

46. Rees:[34]

Man, aged 30, with phthisis. Severe vomiting, intestinal obstruction suspected. Stomach was dilated and filled with fluid. Intestines were collapsed into the pelvis.

47. Robson, Mayo:[35]

Woman: duodeno-choledochotomy performed. Ten days later had severe vomiting with abdominal distension. Death in 24 hours.

Post Mortem: Stomach dilated: adhesions of pylorus to gall bladder and liver.

48. Robson:

Woman, aged 35. Sudden vomiting and epigastric pain after cholecystotomy; stomach dilated, anuria. Lavage. Recovery.

49. Robson:

Woman, aged 29: abdominal hysterectomy. Stomach reached to pubis: succussion obtained. Lavage. Recovery. Stomach normal in a week.

50. Schnitzler:[36]

Boy, aged 17. Acute symptoms after chloroform. Death in three days.

Post Mortem: Stomach and duodenum dilated. Jejunum compressed by superior mesenteric artery.

51. Schultz:[37]

Woman, aged 24, had some vague fever. She appeared to be convalescing when suddenly acute symptoms developed. Stomach was distended and peristaltic waves were visible. Death in three days.

Post Mortem: Stomach distended. No obstruction.

52. Stiles:[38] (personally communicated).

Woman, aged 60: gall-bladder and a portion of transverse colon removed for malignant disease: severe vomiting and distension occurred.

Post Mortem: Stomach filled almost the whole abdomen.

53. Thomson:[39]

Man, aged 48: with chronic dilatation of stomach. After stomach had been washed out there was hiccough, pain, vomiting, collapse, feeble pulse, subnormal temperature, outline of stomach was visible. No rigidity. Death in two days.

Post Mortem: Stomach distended with gas and fluid. Greater curvature reached below level of iliac crest. Pyloric orifice was obstructed by a growth.

54. Thomson:

Man, aged 26, had symptoms of renal calculus. Right kidney explored but no stone found . Vomiting began a few hours after the operation and persisted till death four days later.

Post Mortem: Stomach and first part of duodenum distended with gas and greenish fluid. No obstruction. Marked chronic nephritis.

55. Thomson:

Woman, aged 40, with jaundice. Laparotomy revealed a tumour of pancreas and distended gall bladder. Latter was drained: five days after operation patient had persistent vomiting, scanty urine, collapse, brownish fluid was vomited until death which took place in four days time.

Post Mortem: Stomach distended with gas and fluid. No definite pyloric obstruction.

56. Thomson:

Woman, aged 24, with right pleuro-pneumonia. Had sudden vomiting which continued with a slight interval till death - 36 hours after onset.

Post Mortem: Stomach reached down to pubes. On relieving it of its contents it quickly shrank. No microscopic changes found: left diaphragmatic pleurisy and consolidation of lower lobe of right lung present.

57. Thomson:

Man, aged 37. Sustained an injury to the head and spine: had vomiting for two days and then died.

Post Mortem: Stomach distended with air and contained 8 oz. of fluid. On puncturing its walls it shrivelled up. Duodenum was also dilated: intestines below were moderately collapsed: there was a fracture of the occipital bone and dislocation of 4th and 5th cervical vertebrae and fracture of spinous process of 5th dorsal vertebra.

58. Todd: (40)

Woman, aged 56: had violent pains in the stomach and vomited severely: face was drawn, eyes sunken, hands and feet cold, pulse slow and feeble, succussion obtained. Stomach was dilated and tender. She had eaten a lump of cheese on the previous day and had vomited some of it at night. Stomach tube was passed and gas escaped with some bilious fluid. Recovery occurred. She had had rheumatic fever the year before.

59. Wiesinger: (42)

Man, aged 41, had acute symptoms after an error of diet which suggested obstruction.

Abdomen was opened and the stomach found to be dilated and twisted at an angle of 180°at the pylorus. It was punctured and drained. Recovery was complete.

60. Wright: (43)

Man, aged 22, had symptoms of acute obstruction. Abdomen was opened and stomach found to be dilated; punctured and three pints of fluid withdrawn. Death followed from peritonitis.

Post Mortem: Stomach and duodenum dilated.

61. Wright:

Man, aged 20. Had incessant vomiting, constipation and cramp in muscles: symptoms relieved by lavage and rectal feeding for two days. The condition returned a week later and ended in death.

ANALYSIS OF COLLECTED CASES.

Post Mortem Examination.

Reference No.	Reported by	Sex	Age	Supposed exciting cause	Previous operations (if any)	Previous disease	Result	Time of death after onset of acute symptoms	Parts dilated	Other morbid conditions.
1.	Albrecht	F.	44		Removal of Mamma.	Cancer of breast.	Death.	2 Days.	Stomach and duodenum.	
1.	Albrecht	F.	30		Operation on elbow		Death.	11 Days.	Stomach.	
2.	Albu	M.	26			Scarlet Fever.	Death.	2 Days.	Stomach.	
3.	Appel			Abdominal injury.			Death.		Stomach.	
4.	Bennett	M.	26			Empyema	Death.	9 Days.	Stomach.	
5.	Bettmann	F.	17			Typhoid	Recovery.			
6.	Box & Wallace	M.	29		Amputation of thigh	Cellulitis of knee	Death.		Stomach & 1st & 2nd parts of duodenum.	
6.	Box & Wallace	M.	24			Pleuro-pneumonia.	Death.	3 Days.	Stomach and duodenum.	Left lung pneumonia.
6.	Box & Wallace	M.	16	Blow on epigastrium.			Death.	Few Hours.	Stomach.	
7.	Boas.		20	Error of diet			Recovery			
8.	Broadbent Walter	M.		Error of diet			Death.	2 Days	Stomach.	
9.	Broadbent Wm.	F.	35	Eating an ice			Recovery			
9.	Broadbent Wm.	M.	50	Eating big meal.			Recovery			
10.	Brown H. H.	F.	16			Acute rheumatism and chorea.	Death.	12 Hours	Stomach	Endocarditis.
11.	Brown W. H.	M.	55				Death.	few Hours	Stomach	Nephritis.
12.	Bradshaw	F.					Recovery			
13.	Dickinson.	F.	3	Error of diet		Fatty heart	Death		Stomach	
14.	Dyson.	F.	41	Miscarriage			Death	3 Days.	None obtained.	
15.	Edmunds.	M.	35	Gunshot wound of spine.			Death	9 Hours	Stomach	

Post Mortem Examination.

Reference No.	Reported by	Sex	Age	Supposed exciting cause	Previous operations (if any)	Previous disease.	Result	Time of death after onset of acute symptoms	Parts dilated	Other morbid conditions.
16.	Erdmann	M.	18	Fall			Recovery			
17.	Fagge	M.	30			Phthisis	Death		Stomach	
17.	Fagge	M.	20				Death	4 Hours	Stomach	Retro-duodenal abscess.
18.	Fenger.				Chole-cystotomy.		Death	5 Days	Stomach	
19.	Fisher	F.	35				Death		Stomach and duodenum.	Suppurating thrombus in left cavernous sinus.
19.	Fisher	F.	25			Puerperal fever	Death		Stomach and duodenum	
20.	Frazer	M.	20			Pleuro-pneumonia	Death	2 Days	Stomach	Intestines Full of Fluid.
21.	Furstner	F.	21	Blow on epigastrium			Recovery			
22.	Goodhart	M.	29		Incision of knee.		Death	75 Hours	Stomach	Oedema of Lungs
23.	Hood	F.	19			Necrosis of jaw.	Death	12 Hours	Stomach, duodenum & 6in. of jejunum.	Acute purulent pericarditis.
24.	Hunter	F.	25		Double Salpingo-oophorectomy.		Death	8 Days	Stomach	
25.	Jessop	F.	26	Eating an Apple	Excision of hip		Death	Few Hours	Stomach, duodenum & 6in. of jejunum.	
26.	Kelynack	F.	19			Tuberculous hip.	Death	4 Days	Stomach & 1st part of duodenum.	Right Pleura adherent.
27.	Kemp					Typhoid Fever	Recovery			
27.	Kemp	F.		Overeating			Recovery			
27.	Kemp	F.		Improper diet		Rheumatic Endocarditis	Recovery			
28.	Kirch.	M.	19	Error of diet			Death	2 Days		
29.	Krundat	F.	69				Death	11 Days	Stomach and duodenum.	

Post Mortem Examination.

Reference No.	Reported by	Sex	Age	Supposed exciting cause	Previous operations (if any)	Previous disease.	Result	Time of death after onset of acute symptoms	Parts dilated	Other morbid conditions.
29.	Krundat	M.				Typhoid Fever	Death.	2 Days.	Stomach and duodenum.	
29.	Krundat	M.	22				Death	2 Days.	Stomach and duodenum.	
30.	Lieutaud	M.					Death.		Stomach.	Lungs suppurating.
31.	Meyer	M.	41				Death.	11 Days.	Stomach and duodenum.	
32.	Miller.	F.	48				Death.	10 Days.	Stomach	
33.	Morris.	M.	37			Suppuration of right ankle.	Death.	4 Hours	Stomach	
33.	Morris.	M.			Nephrectomy		Death.	2 Days	none obtained	
	Munro	F.	27			Chlorosis	Death.	15 Hours	Stomach and 1st part of duodenum.	
34.	Rees	M.	30			Phthisis.	Death.		Stomach.	
35.	Robson	F.			Duodeno-choledochotomy		Death.	24 Hours	Stomach	Pyloric adhesions
35.	Robson	F.	35		Cholecystotomy		Recovery			
35.	Robson	F.	29		Abdominal hysterectomy		Recovery			
36.	Schnitzler	M.	17	chloroform anaesthesia.			Death.	3 Days	Stomach and duodenum	Jejunum compressed by mes. artery.
37.	Schultz	F.	24			Fever	Death.	3 Days	Stomach	
38.	Stiles	F.	60		Excision of gall bladder	Carcinoma	Death.		Stomach	
39.	Thomson.	M.	48			Chronic dilatation.	Death.	2 Days	Stomach	Pyloric tumour.
39.	Thomson	M.	26		Nephrotomy		Death.	4 Days	Stomach and 1st part of duodenum.	Nephritis.
39.	Thomson	M.	40		Cholecystotomy	Tumour of Pancreas	Death	5 Days	Stomach	
39.	Thomson	F.	24			Rt. Pleuro-pneumonia.	Death.	36 Hours	Stomach	Pleuro-pneumonia.

Post Mortem Examination

Reference No.	Reported by	Sex	Age	Supposed exciting cause	Previous operations (if any)	Previous disease.	Result	Time of death after onset of acute symptoms	Parts dilated	Other morbid conditions.
39.	Thomson	F.	37			Injury to head and spine.	Death.	2 Days	Stomach and duodenum.	Fracture of occipital.
40.	Todd	F.	56	Error of diet			Recovery			
42.	Wiesinger	M.	41	Error of diet			Recovery			
43.	Wright	M.	22				Death	Few days	Stomach and duodenum.	Peritonitis
43.	Wright	M.	20				Death			

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